OUTCOME OF PERITONEAL DIALYSIS IN THE MANAGEMENT OF ACUTE RENAL FAILURE

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CERTIFICATE

This is to certify that dissertation titled "**OUTCOME OF PERITONEAL DIALYSIS IN THE MANAGEMENT OF ACUTE RENAL FAILURE**" is the bonafide original work of DR. M. JESU ANTONY EZHIL ARASU in partial fulfillment of the requirements for M.D. Branch – I (General Medicine) Examination of the TamilNadu Dr. M.G.R. Medical University to be held in March 2007. The period of study was from March 2005 to July 2006.

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DECLARATION

I **DR. M. JESU ANTONY EZHIL ARASU**, solemnly declare that dissertation titled "**OUTCOME OF PERITONEAL DIALYSIS IN THE MANAGEMENT OF ACUTE RENAL FAILURE**" is a bonafide work done by me at Govt Stanley Medical College and Hospital from march 2005 to July 2006 under the guidance and supervision of my unit chief **PROF. V.RUKMANI, M.D.** Addl. Professor of Medicine.

This dissertation is submitted to the TamilNadu Dr. M.G.R. Medical University, towards partial fulfillment of requirement for the award of **M.D. Degree (Branch -- I) in General Medicine**.

Place: Chennai
Date:

DR.M.JESU ANTONY EZHIL ARASU

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CONTENTS

Introduction

INTRODUCTION

Acute Renal failure is a reversible disorder. It should be detected early and treated promptly by correction or removal of the precipitating factor. Management may include conservative measures or dialysis depending on the severity of the renal impairment and resulting complications.

Haemodialysis necessitates constant attendance of the trained professional and technical personnel, costly equipment heparinisation of the patients and surgical placement of arterial and venous catheters. At least two or three hours of preparations are needed before starting the dialysis, so its usage and utility is restricted even in the secondary centers.

Peritoneal dialysis is a simple, safe, effective and relatively less expensive and alternative procedure in renal failure, especially acute renal failure. It does not require expensive instruments and trained technicians and can be performed by anyone at any center which can save many lives.

With the above considerations, the present study was undertaken to evaluate the efficiency and to record the complications of intermittent peritoneal dialysis in the management of acute renal failure.

Aim of study

AIMS OF THE STUDY

1. To study the age and sex distribution, various clinical presentation of acute renal failure.

2. To analyze the various etiologies of acute of renal failure.

3. To analyze the complications of peritoneal dialysis

4. To assess the therapeutic outcome and efficacy following peritoneal dialysis

Review of literature

REVIEW OF LITERATURE

HISTORICAL REVIEW

IN 1744, The original concept of peritoneal dialysis stems from Reverem Stepen Hales.

IN 1877, The result of an animal experiment by perfusing abdominal cavity of rabbit with cold saline and observing the decrease of body temperature was published by a German investigation 'WEGNER'[1]

IN 1918, Black-Tan and Maxcy, American pediatricians were first to utilize peritoneal cavity for administration of fluid to dehydrated children[4].

IN 1923, Ganter professor of Medicine at Wuzeburg, Germany, first attempted peritoneal dialysis in a human being. He instilled 1.5 Liter physiological saline in peritoneal cavity of the patient who becomes acutely uremic following bilateral ureteric obstruction due to malignancy which resulted in transient improvement.

IN 1937, Housser and Werder modified Ganter's techniques by inserting two catheters in the abdomen. One for in flow and other for out flow.

IN 19571, Grollman was the first man to introduce intermittent peritoneal lavage which is the basis of technique used today[2].

IN 1959, Maxwell and associates introduced standard commercially prepared solutions with sterile tube of administrations; they introduced flexible nylon catheter which can be easily introduced through the linea Alba with trochar[3].

Dr. Wester and Roberts made a simple modification by inserting a pointed stylet within a Maxwell catheter, thus eliminating the need for a trochar and sutures[4].

Doolan and his co-workers, Maxwell and associates contributed much to the improvement and perfection of techniques in peritoneal dialysis by hanging bottle system in late fifties and early sixties.

IN 1968, Tenchkoff catheter was introduced which was a major advance for intermittent peritoneal dialysis, and is still widely used. Now peritoneal dialysis evolved in to simple, machine free, power free natural member mediated replacement of renal function.

ACUTE RENAL FAILURE

ACUTE RENAL FAILURE

Acute Renal Failure is a syndrome characterized by rapid (Hours to weeks), decline in GFR and retention of nitrogenous waste products such as blood urea nitrogen and creatinine

For purpose of diagnosis and management, ARF is conveniently divided into three categories.

CLASSIFICATION AND MAJOR DISEASE CATEGORIES CAUSING ACUTE RENAL FAILURE

DISEASE CATEGORY	PERCENTAGE OF PATIENTS
1. Prerenal azotemia caused by acute renal hypoperfusion	55 -- 60
2. Intrinsic renal azotemia caused by acute disease of renal parenchyma	35 -- 40
a. Disease involving large renal vessels	
b. Disease of small renal vessels and glomeruli	

c. Acute injury to renal tubule

   mediated by ischemia or toxins (Accounts for

   more than 90% of the cases in the intrinsic

   renal azotemic category in most series)[5,6].

d. Acute disease of the tubulointerstitium

3. Post renal azotemia caused by acute

   Obstruction of urinary collecting system.

The diagnosis of ARF Usually hinges on serial analysis of BUN and Serum creatinine; though these are relatively insensitive indices of Glomerular filtration and several caveats must be entertained when extrapolating their levels of GFR. GFR may fall by 50% before the serum creatinine value rises because the initial decrement in creatinine filtration is matched by enhanced creatinine secretion by proximal tubule cells.

ETIOLOGY OF ARF

PRERENAL AZOTEMIA

Prerenal azotemia is the most common cause of ARF and is an appropriate physiologic response to renal hypoperfusion[7,8].

By definition, the integrity of renal parenchymal tissue is maintained and GFR is corrected rapidly on restoration of renal perfusion and Glomerular ultrafiltration pressure.

True or effective hypovolemia leads to a fall in a mean systemic arterial pressure, which in turn triggers arterial (eg.carotid sinus) and cardiac baroreceptors and initiates a series of neural and humoral responses that include activation of the sympathetic nervous system and renin- angiotensin- aldosterone system and release of antidiuretic hormone. [9,10,11,12] Nor epinephrine, angiotensin II and ADH act in concert in an attempt to preserve cardiac and cerebral perfusion by stimulating vasoconstriction in relatively non-essential vascular beds such as the musculocutaneous and splanchnic circulations, inhibiting salt loss through sweat glands, stimulating thirst and salt appetite and promoting renal salt and water retention.

Glomerular perfusion, ultra filtration pressure and filtration rate are preserved during mild hypo perfusion through several compensatory mechanisms[13].

Stretch receptors in the afferent arterioles in response to a reduction to perfusion pressure trigger relaxation of afferent arteriolar smooth muscles cells and vasodilatation (auto regulation) Intrarenal Biosyntesis of vasodilator prostaglandin (eg.Postacyclin), Prostaglandin B2) kallikrain and kinins and possible nitric oxide (No) is enhanced.[14 - 19]

Angiotensin II may induce preferential constriction of efferent arterioles possibly by virtue of the increased density of angiotensin II receptors in this location[20]. As a result renal plasma that is filtered by Glomerul filtration fraction is increased and GFR is maintained. These compensatory renal responses are overwhelmed during states of severe hypo perfusion and ARF ensues[13]. Auto regulatory dilation of afferent arterioles is maximal at a mean systemic arterial blood pressure of about 80mm Hg and hypo perfusion below this level is associated with a precipitous decline in Glomerular ultra filtration pressure and GFR.

INTRINSIC RENAL AZOTEMIA

DISEASES OF LARGE RENAL VESSELS.

Occlusion of large renal vessels, either arteries or veins, is an uncommon cause of ARF. To affect BUN and serum creatinine, occlusion must be either bilateral or unilateral in patients with underlying chronic renal insufficiency or solitary functioning kidney.

Renal artery may be occluded acutely by atheroemboli, thrombosis, dissection of aortic aneurysm, or rarely vasculitis. Atheroemboli is the most common culprits and are usually dislodged from an atheromatous aorta during arteriography, angioplasty or aortic surgery. [21,22]

Cholesterol emboli lodge in medium or small renal arteries, where they incite an inflammatory reaction characterized classically be intimal proliferation of vessel wall by macrophages and giant cells, fibrosis and irreversible occlusion of the vessel lumen. [23]

Thrombo emboli arise most commonly from the heart in patients with atrial arrythmias and produce acute infarction of renal tissue[24, 25, 26].

Renal vein thrombosis is an extremely rare cause of ARF and is usually encountered as a complication of nephrotic syndrome in adults or severe dehydration in Children[27, 28, 29].

DISEASE OF MICRO VASCULATURE.

Virtually all diseases that compromise blood flow within renal microvasculature may induce ARF. These include

Inflammatory (eg. Glomerulonephritis and vasculitis)

Non-inflammatory (eg.malignant Hypertension)

Diseases of the vessel wall

Thrombotic microangiopathies

Hyperviscosity Syndromes

Indeed, the decrement in renal perfusion in these settings may be severe enough to trigger superimposed ischemic ATN[30]

Disorders of the tubulointerstitium that induce ARF, other than ischemia or tubular cell toxins include allergic Interstitial nephritis, severe infections, allograft rejection and rarely infiltrative disorders such as sarcoid, lymphoma and leukemia.

ACUTE TUBULAR NECROSIS

ISCHEMIC ACUTE TUBULAR NECROSIS

Prerenal azotemia and ischemic ATN are part of a spectrum of manifestations of renal hypoperfusion, prerenal azotemia being a response to mild to moderate hypoperfusion and ischemic ATN being the results of more severe or prolonged hypoperfusion often coexist with other renal insults (eg.nephrotoxins or sepsis). ATN is associated with injury to renal parenchyma and does not resolve immediately on restoration of renal perfusion.

NEPHROTOXIC ACUTE TUBULAR NECROSIS

It complicates the administration of many structurally diverse pharmacologic agents and poisons[31]. In General nephrotoxins cause renal injury by inducing a varying combination of intrarenal vasoconstriction, tubule toxicity, and or intratubular obstruction. The kidney is particulary vulnerable to nephrotoxic renal injury by virtue of its rich blood supply (25% of cardiac output) and its ability to concentrate toxins to high levels within the medullary interstitium (via the renal counter current mechanism and renal epithelial cells (via specific transporters) [31].

In addition the kidney is an important site for xenobiotic metabolism and may transform parent compounds into relatively harmless metabolites [31].

The nephrotoxic potential of most agents is dramatically increased in the presence of border line or overt renal ischemia, sepsis or other insults.

POST RENAL AZOTEMIA

Urinary tract obstruction accounts for less than 5% of the cases. ARF resulting from obstruction requires either obstruction or urine flow between the external urethral meatus and bladder neck. Bilateral ureteric obstruction, or unilateral ureteric obstruction in a patient with one functioning Kidney or underlying chronic renal insufficiency.

During the early stages of the obstruction (hours to Days) continued glomerular filtration leads to increased intraluminal pressure upstream of the site of obstruction. This results in gradual distension of proximal ureter, renal pelvis, and calyces and a fall in GFR.

Although acute obstruction may lead to an initial modest increase in renal blood flow arterial vasoconstriction soon supervenes, leading to further decline in Glomerular filtration.

PATHOPHYSIOLOGY OF ISCHEMIC ARF

The cause of Ischemic ARF is typically characterized by three phases.

1. Initiation phase (hours to days)
2. Maintenance phase (1 to 2 weeks)
3. Recovery phase

INITIATION PHASE

In this phase GFR declines because

a. Glomerular ultrafiltration pressure is reduced as a consequence of the fall in renal blood flow.

b. The flow of glomerular filtrate within tubules is obstructed by casts comprising epithelial cells and necrotic debris derived from ischemic tubular epithelium

There is back leak of glomerular filtrate through injured tubular epithelium.

Ischemic injury is most prominent in the terminal medullary portion of the proximal tubular segment, pars-recta and the medullary portion of the thick ascending limb of the loop of henle.

2. MAINTENANCE PHASE

The initiation phase is followed by a maintenance phase during which epithelial cell injury is established, GFR stabilities at its nadir (typically 5 to 10 ml/min) urine output is lowest and uremic complications arise

Persistent intrarenal vasoconstriction, medullary ischemia triggered by dysregulated release of vasoactive mediators from injured endothelial cells, (eg. Decreased nitric oxide, increased endothelin) congestion of medullary blood vessels, and reperfusion injury induced by reactive oxygen species and other mediators derived from leukocytes damages renal parenchymal cells.

In addition, epithelial cell injury per se may contribute to persisitant intrarenal vasoconstriction by a process termed tubuloglomerular feed back.

3. RECOVERY PHASE

It is characterized by tubule cell regeneration and a gradual return of GFR towards premorbid levels. The recovery phase may be complicated by marked diuresis. Diuretic phase is due to excretion of retained salt and water and other solutes, continued use of diuretics and delayed recovery of epithelial cell function (solute and water reabsorption) relative to Glomerular filtration.

CONFIRMATORY TESTS

The pattern of change in serum creatinine value often provides clues to the cause of ARF

Serum creatinine rise

Within 24 to 48 hrs - in renal ischemia due to

1. atheroembolisation
2. radiocontrast exposure

- Usually peaks after 3-5 d and return to normal range in 5-7 d in contrast nephropathy.
- Usually peaks after 7-10 d and returns to normal in 10-14 d in ischemic ATN
- Usually peaks after 7-10 d and frequently irreversible in atheroembolic disease.

Imaging of urinary tract by plain film or abdomen ultrasonography, computed tomography or magnetic resonance is recommended for most patients with ARF to distinguish between acute and chronic renal failure and exclude acute obstructive uropathy[32].

Plain film of abdomen with tomography if necessary usually provides a reliable index of kidney size and may detect Ca ++ containing kidney stones.

However the capacity of ultrasonography to determine cortical thickness, differences in cortical and medullary density and the integrity of the collecting system in addition to kidney size, makes it the screening modality of choice in most cases of ARF[33].

Renal biopsy is useful when cause of intrinsic renal azotemia is unclear.

MANAGEMENT

Because there are no specific therapies for treatment of ischemic and nephrotoxic ARF prevention is of paramount importance.

SPECIFIC THERAPIES

- Prerenal azotemia is rapidly reversible upon correction of primary hemodynamic abnormality.

- Postrenal azotemia – resolves upon relief of obstruction.

- No specific therapies for ischemic or nephrotoxic ARF. Management of these disorders should foucus on elimination of the causative hemodynamic abnormality, toxin, avoidance of additional insults and prevention and treatment of complications.

- Specific treatment of other causes of intrinsic renal azotemia depends on the underlying pathology,

Dialysis replaces renal function until regeneration and repair restore renal function. Hemodialysis and peritoneal dialysis appear equally effective for management of ARF.

PERITONEAL DIALYSIS

Peritoneal dialysis has become a simple procedure which is now available in most hospitals [35, 36, 37.] It is done with commercially prepared solutions and a disposable catheter[38] is easy to set up and carries an low incidence of complications.

STRUCTURE AND FUNCTION OF THE PERITONEAL MEMBRANE.

A Monocellular layer of membrane lines the inner surface of the abdominal wall and reflects over the visceral organs, covering them and creating a space of the peritoneum.

When aqueous solutions are instilled into the peritoneal cavity their solute composition approaches Gibbs Donnan equilibrium with plasma by diffusion along the electrochemical concentration gradients. This is the major mechanism of solute transfer during peritoneal dialysis and the rationale underlying its clinical use. Concurrently iso-osmotic solutions are absorbed into the circulation predominantly through the diaphragmatic lymphatics at a rate faster than the rate of ultrafiltration, which is induced by transcapillary hydrostatic pressure gradient alone. To achieve net ultra filtration, a slowly or non-diffusible solute must be added to the solution instilled into the peritoneum to serve as an osmotic agent. These two process, diffusion and osmotic ultrafiltration, govern peritoneal dialysis, although net solute and water removal are reduced by the absorption of solution through lymphatics. Solutes such as fatty acids typically enter peritoneal

dialysate through metabolic production where as others may undergo hepatic biotransformation during absorption.

The volume of peritoneal dialysis solution per exchange is typically only about 5% of the total body water, Hence solute equilibration between plasma water and peritoneal dialysate occurs rather quickly. As a consequence, in order to achieve maximal transport per minute, the dialysate must be exchanged reasonably rapidly, despite a relatively low blood flow to the peritoneum.

The peritoneal diffusion barrier consists of fluid films in the blood and in the dialysate, the endothelium, the mesothelium their basement membranes and the intervening interstitium[39].

There is evidence favoring the concept that during peritoneal dialysis solutes transfer through intercellular channels. Transcellular transport is not considered quantitatively important. It is noteworthy that dialysate potassium and sodium equilibrate with the concentrations in the extra cellular fluid, not with concentrations within cells[40].

The diffusion of large solutes is limited predominantly by the dimensions of the intercellular pores of the expillary wall but movement of the solutes is restricted by stagnant fluid films of the blood, dialysate and interstitium. The blood flow rate and dialysis fluid volume and exchange rate select these fluid lines to a great extent. Higher peritoneal blood flow rates are ordinarily accompanied by higher

blood volume, which should increase capillary for diffusion and also accelerating transport of solutes.

GENERAL PRINCIPLES

The peritoneal cavity is lined by a semipermeable membrance which has an area of about 2.2 square meters and its solutes are transferred across the membrane, mainly by diffusion.

The clearance of a solute such as urea depends on a number of factors.

EQUILIBRIUM TIME

When fluid is introduced into the peritoneal cavity, diffusion occurs rapidly for the first 30 min and than more slowly for the following 2 hrs.

RATE OF DIALYSATE FLOW

When the flow of the dialysing fluid is increased from 1 litre to 3.5 litres / hours, urea clearance increases from 12 ml / min to 30 ml / min. Further increments in clearance may be obtained with higher flow rates[41].

TEMPERATURE

Heating dialysis fluid from 20 to 37C causes dilatation of peritoneal vessels and increases urea clearance by 35%[42].

DIALYZING FLUID

Hypertonic solutions increase urea transport because of solvent drag and increased permeability of the peritoneal membrane. The use of 7% instead of 1.5% glucose concentration in the dialysis fluid increases urea clearance by about 50%[43].

PERMEABILITY OF THE PERITONEUM

This increased by heat, hypertonic solutions, infections and experimentally by surface active agents such as dioctyl sodium sulfo succinate, Decreased permitability to creatinine, urate and other larger molecules may occur in the acute renal failure associated with heat stress and exercise[44].

INDICATIONS FOR PERITONEAL DIALYSIS[45]

- Anuria or Oliguria more than 24 Hours.
- Hyperkalemia (definitely if > 6.5 meq/l)
- Pulmonary Edema
- Metabolic acidosis
- Uremic Encephalopathy
- Uremic Pericarditis
- Uremic Syndrome

- Blood area > 200mg/dl

- Serum creatinine > 10 mg/dl

COMPOSITION OF DIALYSATE FLUID

The concentrations of Dextrose used are 1.5%, 2.5%, 3.5% or 4.25, 7%. The solution generally used is concentrations with 1.5% and 4.25% dextrose fluid. They can be used to remove excess of body water.

- Sodium concentration – 132 meq/l

- Magnesium concentration varies from 0.5 to 1.5 meq/l

- Calcium concentration varies from 2.5 to 3.5 meq/l

- Chloride Concentration varies from 95 to 102 meq/l

- Lactate Concentration Varies from 35 to 40 meq/l

PROCEDURE:

Peritoneal dialysis is usually done with commercially available administration sets and solutions and with a disposable thin stylet cannula which is inserted percutaneously[46].

The patient's bladder is emptied and the patient is weighed. The cannula is inserted under local anaesthesia in a midline site about 2-3 inches below the umblicus It is helpful if the patient raises the head at the moment of insertion to tighten the abdominal muscles and prevent perforation of the bowel. A feeling of 'give' indicates that the peritoneal cavity has been entered. The stylet is then removed. The cannula is advanced towards the left pelvic gutter, fixed with sutures and tape and connected to the dialysis bottles through 'Y' tube set.

Two litres of warmed dialysate are run into the peritoneal cavity, allowed to remain for 30 min and drained out. The entire cycle is conveniently timed to last one hour for a more rapid dialysis the equilibration time may be shortened so that the cycle is reduced to 45 or even 30 minutes[47]. However this requires more fluid and increases costs. Dialysis time is usually limited to 48 hours. The cannula is then removed but may be reinserted later if necessary.

A dialysis, fluid potassium concentration of 2 meq/l is generally used but in hyperkalemic patients potassium should be omitted for about 10 exchanges.

In digitalized patients, various arrythmias may occur if the potassium level is

lowered too quickly and potassium concentration of 4 meq/l is therefore recommended.

Hypertonic solutions (4.5 to 7% glucose) may be used to remove fluid in overhydrated patients. A careful record must be kept of the patient's weight and the amount of fluid exchanged to prevent hypovolemia.

During dialysis, the patient usually takes an unrestricted diet and is encouraged to sit in a chair.

PERITONEAL DIALYSIS – ADVANTAGES:

Peritoneal dialysis is a simpler technique doesn't require qualified personnel and it is safe due to the absence of extra corporeal circuit.[47]

Peritoneal dialysis avoids systemic heparinisation making it most desirable in treating patients in the immediate post operative period with severe trauma and recent wound.[48]

It is preferred in some medical conditions like intracerebral haemorrhage and hyper coagulable state where again heparin is contraindicted. [49]

In Haemodialysis rapid correction of biochemical abnormalities of uremia will cause disequilibrium syndrome which will not occur in peritoneal dialysis.[50]

Peritoneal dialysis is useful in hypotensive patients in whom perfusion of the

peritoneal membrane is still adequate where haemodialysis has its own risk.

Peritoneal dialysis has provided convenient and satisfactory therapy for the pediatric patients.[51] Access for the vascular cannulation for haemodialysis is a problem in children, again selection of appropriate size of haemodialysis is another problem in paediatric patients where peritoneal dialysis is an alternative[49].

In paediatric age group peritoneal dialysis provides easy access to peritoneal cavity and peritoneal surface area is larger, calculation for the amount of dialysate to be instilled is very simple (eg.50mil/kg for infants; 28-35ml/kg for 3-8yrs).[49]

Peritoneal dialysis has excellent cardiovascular tolerance. It is beneficial in elderly patients also in patients with cardiovascular problems.

In Diabetic patients simultaneous administration of the intraperitoneal insulin along with dialysate provide adequate control which favours usage in diabetes.

HIV, Hepatitis B and C infection are common in patients undergoing haemodialysis which does not occur in peritoneal dialysis because it does not involve extracorporeal circulation.

PERITONEAL DIALYSIS-LIMITATIONS.

It has its own limitations

Peritoneal dialysis has limited utility for the treatment of hypercatabolic states like trauma patients or surgical patients who tend to have rapidly increasing plasma concentration of potassium, urea and posphate.[52]

The usefulness is limited because of long duration for dialysis usually 24-36 hours in single sitting.

Peritoneal dialysis has relative inefficiency in removing potassium and toxic substance[52].

Peritoneal dialysis may be difficult in post operative patients with multiple abdominal drains, post operative adhesions, infection, abdominal trauma, severe Gastero esophageal reflux, and in intestinal ileus.

Peritoneal dialysis has also caused abdominal pain or discomfort during dialysis Bleeding or leakage fluid around the puncture site, catheter malfunction, peritonitis. Rare complications like Bowel and Bladder injury, atelectasis, pneumonia and purulent bronchitis[53]. Hypovolemia or hypervolemia, metabolic complications like hyperglycemia, hypokalemia, hyponateremia and lactic acidosis.

Materials and methods

MATERIALS AND METHODS

The study was conducted on 65 patients of acute renal failure admitted in the medical and nephrology wards of the Govt. stanley medical college Hospital for treatment from November 2005 to July 2006.

All the patients were thoroughly evaluated by detailed history, clinical examination, biochemical parameters, ultrasonogram, peritoneal fluid cell count and culture as per preformed proforma.

Various pre and post dialysis investigations included the following (1) total and differential count, haemoglobin, (2) clotting time (3) Blood urea (4) Serum creatinine (5) Blood sugar (6) Serum electrolytes (7) urine analysis. (8) X-ray chest and abdomen.

SELECTION CRITERIA:

The following criteria were used for selection of patients with acute renal failure.

1. Rapid reduction of Glomerular filteration rate leading to sudden and progressive elevation of Blood Urea > 40 mg/dl and or serum creatinine >1.5 mg/dl.

2. Normal sized or Enlarged Kidney revealed by ultra sonogram.

3. Absence of pre existing renal disease.

EXCLUSION CRITERIA:

1. The patients with Bilateral contracted kidneys on ultrasonogram.

DIAGNOSTIC CRITERIA FOR PERITONITIS.

At least two of the three following conditions should be present.

- Symptoms and signs of peritoneal inflammation
- Cloudy peritoneal fluid with an elevated peritoneal fluid count (>100/ul) due to predominantly (>50%) neutrophils.
- Demonstration of bacteria in the peritoneal fluid by Gramstain or culture.

METHODOLOGY ADOPTED IN PERITONEAL DIALYSIS:

The total of 84 procedures of intermittent peritoneal dialysis were performed and the frequency being 1 to 3 per person with duration of 24 hours per sitting.

PROCEDURE:

MATERIAL REQUIRED

- Disposable syringes with needle.
- Mosquito artery forceps.
- Sponge holding forceps.
- Iodine solution
- Sterile dressing
- 2% Lignocaine
- Peritoneal dialysis catheter set
- Peritoneal dialysis infusin set
- Needle holder with half circle cutting needle.
- Thread and adhesive tape
- Drip stand with two hangers
- Heparin

Before starting the peritonical dialysis, sample for Blood urea, sugar and serum creatinine and Electrolytes were taken. The patients Bladder is emptied and he is

weighed. Abdomen is prepared with savlon followed by iodine and spirit. Wrapping with sterile towel is done all around umbilicus, leaving small Infra – umbilical area open. Midline local anaesthesia (2%) lignocaine – 3 to 5 ml) is given one-inch below umbilicus upto parietal peritoneum. PD catheter and infusion set were opened and set was connected to the 2PD bottles.

An Incision was made one inch below umbilicus upto parietal peritoneum. PD catheter is inserted through the incision. A feeling of “give” indicates that peritoneal cavity has been entered. The stylet of catheter is withdrawn little out so that sharp edge come inside the cannula and trauma to the intra abdominal structures will be minimal. The catheter is advanced towards the pouch of Douglas and the catheter fixed with sutures and connected to the dialysate bottle through Y tubing.

If the calculated (35 – 50ml/kg) fluid has gone into the abdomen, the dialysate is drained immediately. Again the calculated dialysate is instilled into the peritoneal cavity and allowed for 30 minutes to stay and them drained out. This one exchange, likewise it is done for 24 exchanges. During the entire peritoneal dialysis a balance chart is maintained stating the amount of the dialysate instilled into the peritoneal cavity and the amount of dialysate drained at the end of each exchange.

At the end of the peritoneal dialysis, dialysate, fluid from abdomen was sent for

cell count, culture and sensitivity.

POST PERITONEAL DIALYSIS FOLLOWUP:

After the peritoneal dialysis Blood Urea, Blood Sugar, Serum creatinine, Serum electrolytes were estimated.

Daily blood urea, serum creatinine and serum electrolytes were done after the peritoneal dialysis, till clinical and biochemical parameters to become normal.

Results and observations

RESULTS AND OBSERVATIONS

TABLE I

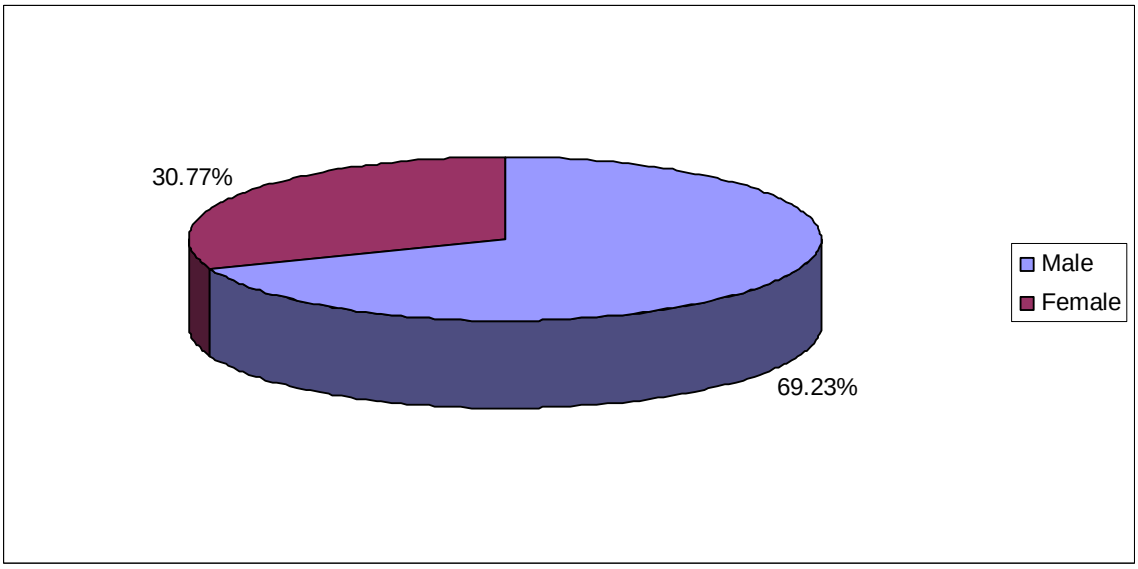
AGE SEX DISTRIBUTION

TABULAR VIEW

Age in Years	Males	Females	Total	Percentage
<20	4	0	4	6.67
20 -- 40	27	7	34	56.67
41 -- 60	13	7	20	33.33
>60	1	1	2	3.33
Total	45 (75%)	15(25%)	60	100

AGE SEX DISTRIBUTION

GRAPHICAL VIEW



AGE SEX DISTRIBUTION

GRAPHICAL VIEW

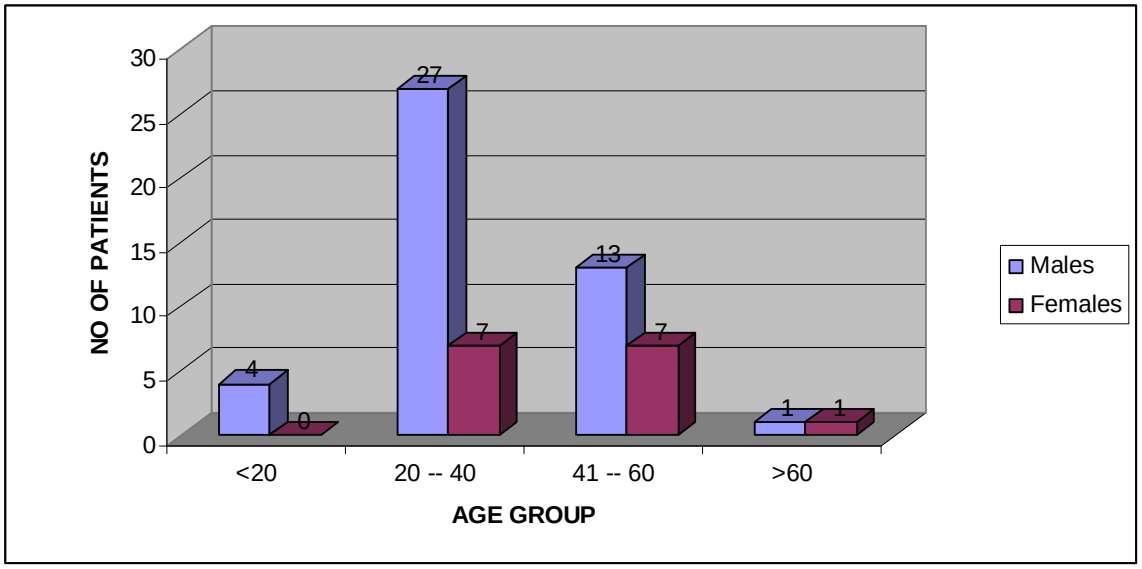


TABLE II

ETIOLOGY ACUTE RENAL FAILURE

TABULAR VIEW

Etiology Of ARF	Males	Females	Total	Percentage
Malaria	1	1	2	3.33
Snake Bite	4	1	5	8.33
CuSO4	10	1	11	18.33
AGE	15	6	21	35
AGN	8	Nil	8	13.33
Septiceamia	3	Nil	3	5
Leptospirosis	2	3	5	8.33
Drug Induced	2	Nil	2	3.33
Obstetric	Nil	3	3	5

ETIOLOGY ACUTE RENAL FAILURE

GRAPHICAL VIEW

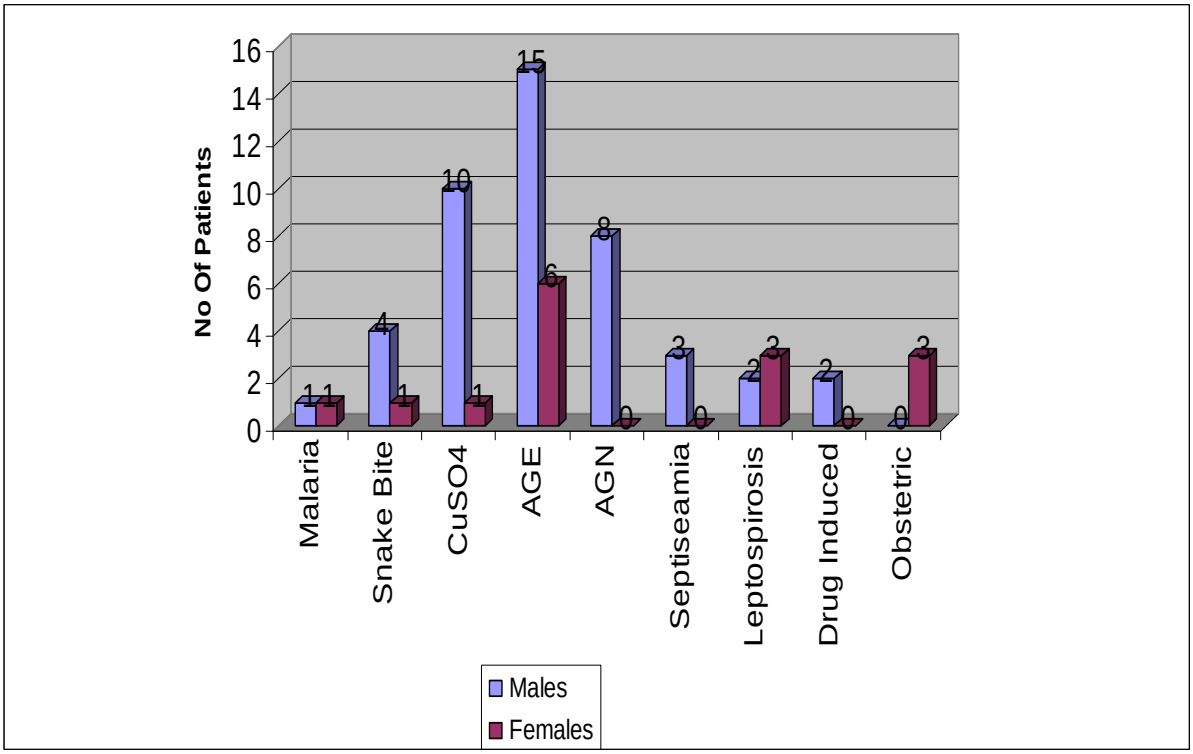


TABLE III

SYMPTOMS AND SIGNS – AT THE TIME OF PRESENTATION

	Males	Females	Total	Percentage
Anuria /oliguria	45	15	60	100
Heamorrhage	10	2	12	20
Metabolic Acidosis	6	1	2	11.67
Putmonary edema	Nil	Nil	Nil	Nil

TABLE III

SYMPTOMS AND SIGNS – AT THE TIME OF PRESENTATION

GRAPHICAL VIEW

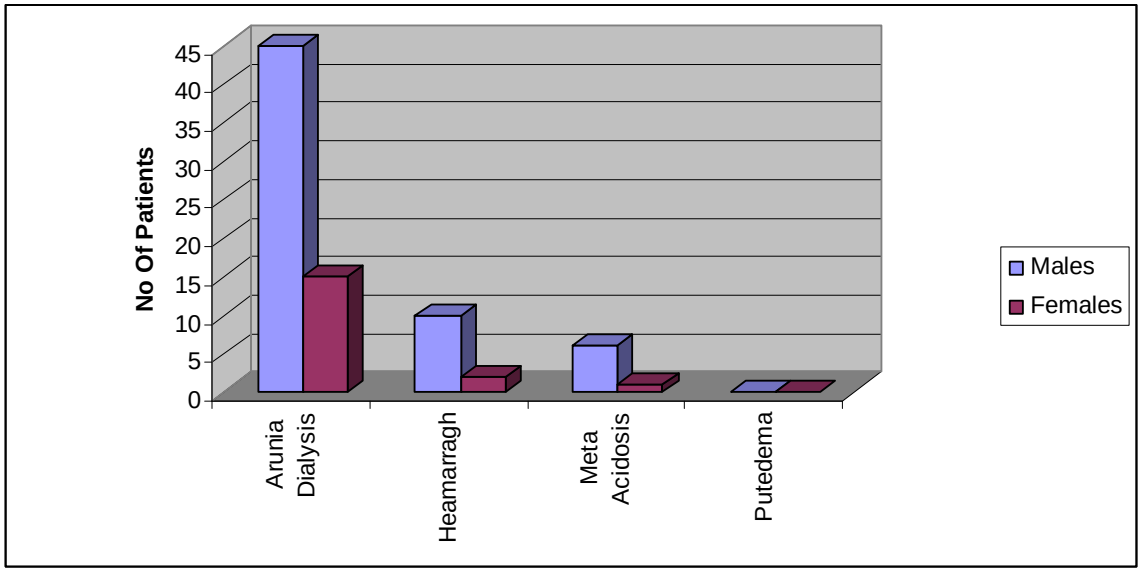


TABLE IV

BIOCHEMICAL CHANGES BEFORE AND AFTER PERITONEAL DIALYSIS

	Average Pre-Dialysis	Average Post-Dialysis	Change
Urea	106.05	34.74	71.31
Creatinine	6.225	1.47	4.755

TABLE V

COMPLICATIONS OF PERITONEAL DIALYSIS

Complications of Peritoneal Dialysis	Number of Patients	Percentage
Heamorrhage in Peritoneal Cavity	10	16.67
Mild	7	11.67
Severe	3	5
Peritonitis	Nil	nil
Abdominal Pain	34	56.67
Catheter Block	16	26.67
Perforation of Gut	Nil	nil

TABLE V

COMPLICATIONS OF PERITONEAL DIALYSIS

GRAPHICAL VIEW

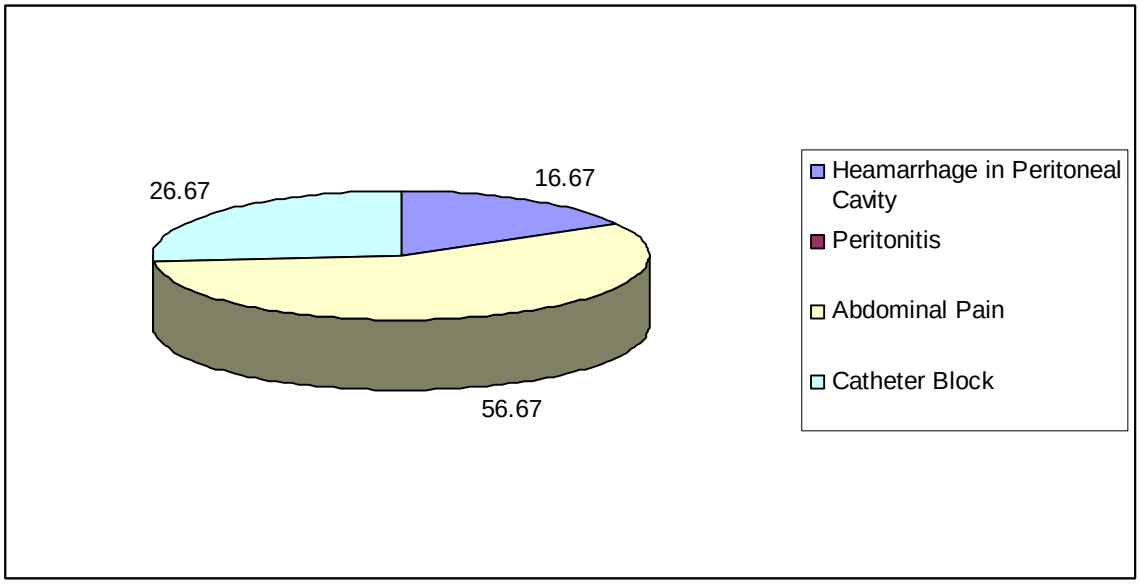


TABLE VI

DISTRIBUTION OF 60 PATIENTS WITH ACUTE RENAL FAILURE ON THE BASIS OF SERUM CREATININE

		Group I (<4 mg/dl)	Group II (4-7 mg/dl)	Group III (>7 mg/dl)
Number of Patients	Total	7	33	20
	Males	4	27	14
	Females	3	6	6
Mean Age (Years)	Total	41.14	38.03	37.5
	Males	35.25	37.15	34.21
	Females	49	42	45.17
Mean Creatinine		3.14	5.3	8.6
Mean Number of PD Per Patient		1.29	1.39	1.45
Mean Urea Concentration		75.43	103.72	125.5
Mortality Rate		Nil	18.51%	20%

TABLE VII

OUTCOME

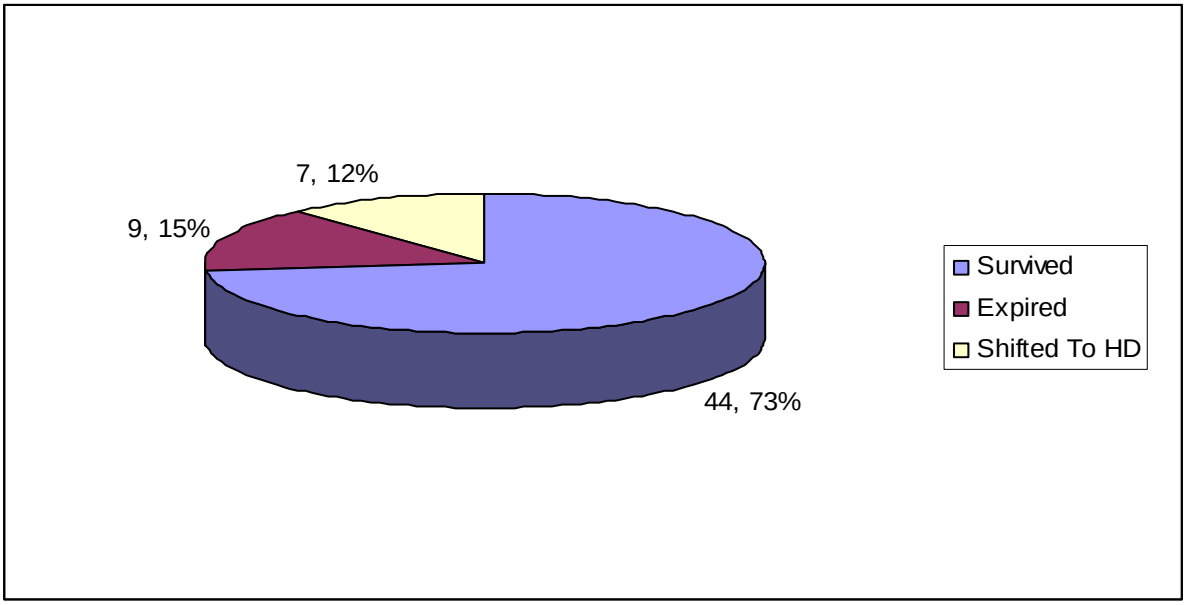
TABULAR VIEW

Etiology Of ARF	Total	Group I (S)	Group II (Ex)	Group III (HD)
Malaria	2	1	1	Nil
Snake Bite	5	5	Nil	Nil
CuSO4	11	7	1	3
AGE	21	17	3	1
AGN	8	5	3	Nil
Septiceamia	3	2	1	Nil
Leptospirosis	5	3	Nil	2
Drug Induced	2	1	Nil	1
Obstetric	3	3	Nil	Nil
Total	60	44 (73.33%)	9 (15%)	7 (11.67 %)

TABLE VII

OUTCOME

GRAPHICAL VIEW



Analysis and discussions

ANALYSIS AND DISCUSSION

AGE AND SEX INCIDENCE

In this study, the age of the patient varies from 15years to 65 years. The study group comprised 45 males and 15 females (75% and 25% respectively)

Majority of patients belonged to age group of 20-40 years (56.67%). In the study by R.G.Singh etal (1982) the majority of patients for above mentioned age group was 66.66%.

The predominance of the males over the females is similar to other reported series (Rai, loc, cit; zilleruelo et al, 1978).[54]

However, Holiday et al (1971) did not find any significant sex difference.[55]

ETIOLOGY

In this study the most common etiology for Acute renal failure is acute gastro enteritis in 21 patients (35%) followed by copper sulphate in 11 patients (18.33%) Acute Glomerulonephrites in 8 patients (13.33%) Leptospirosis and Snake Bite each 5 patients (8.33 %) septicaemia and obstetric causes each 3 patients (5%) and Malaria and drug induced in each 2 patients (3.33%) and peritoneal dialysis is not influenced by Biology of Acute renal failure statistically ($P>0.05.Z=0.34$)- TABLE-VII

SYMPTONS AND SIGNS

In this study, oliguria is the commonest symptom present in 60 patients (100%) studies by R.G. Singh et al (1982) Showed oliguria in 95.2%

In this study, Hematuria in 12 patients (20 %) studies by R.G.Singh et al (1982) showed incidence of Hematuria in 52.3%

In this study, 7 patients had clinically metabolic acidosis (11.67 %) study by R.G. Singh et al (1982) showed incidence of metabolic acidosis in 24%

In this study, No patients had pulmonary edema, Study by R.G.Singh etal (1982) showed incidence of pulmonary edema in 14.4 %.

BIOCHEMICAL CHANGES AFTER DIALYSIS.

In this study, the average predialysis value of urea is 106.05mg% and average post dialysis value of urea is 34.74 mg% and change is 71.31mg%. The average pre-dialysis value of creatinine is 6.23mg% and the average post dialysis value of creatinine is 1.47mg%. The change is 4.76mg%.

The study by Acharya et al (1976)56 showed reduction in mean blood urea of 53.9 mg% and reduction in mean serum creatinine of 2.3mg%

Statiscally There is no significant relationship between rising level of Serum

creatinine and mortality in patients with Aute renal failure treated by IPD (P>0.05Z=2.4) – TableVI.

INCIDENCE OF COMPLICATIONS.

In this study 3 patients had severe and 7 patients had mild haemorrhage in the peritoneal cavitiy (16.67%)

Study by K.S.Chugh et al (1975) [57] showed incidence of Haemorrhage in peritoneal cavity as 22.1% R.G.Singh et al(1982) found in 10% of the patients.

Peritonitis was evidenced clinically in none of the patients

Study by K.S.Chugh et al showed incidence of peritonitis as 4 percent.

No peritonists was noticed in study by R.G.Singh et al.

Study by EP. Simkin et al (1968) showed incidence of perforation of gut as 0-1-1.3%

Study by K.S.Chugh et al showed incidence of perforation of gut as 1.16%. There is no perforation of gut in our study. In our study, the commonest complication is Abdominal pain and discomfort which is statistically highly significant (P<0.001, Z=7.67) - Table-V

OUTCOME:

In this study, overall survival rate was 73.3% (44 out of 60 patients) overall mortality rate was 15% (9 out of 60 patients), 7 patients (11.67%) had shifted from peritoneal dialysis to haemodialysis because of their hypercatabolic state and relatively slow response to peritoneal dialysis. Six of them showed complete recovery, one patient expired. Patients who had serum creatinine level more than 7mg/dl had highest mortality rate of 20%

Study by R.G.Singh etal showed incidence of overall survival rate of 71.42%

Study by K.S.Chugh et al showed overall survival rate of 55%

Overall Survival rate of patients in our study in comparison to study by K.S.CHUGH et al is statistically highly significant ($P<0.001$.$Z= 3.11$)

Conclusions

CONCLUSION

1. MAJORITY OF PATIENTS OF ACUTE RENAL FAILURE BELONG TO THE AGE GROUP OF 20-40 YEARS.

2. THE INCIDENCE OF ARF WAS HIGHER IN MALES THAN FEMALES.

3. IN THIS SERIES MOST COMMON ETIOLOGY OF ACUTE RENAL FAILURE IS ACUTE GASTRO ENTERITIS.

4. THE MOST COMMON PRESENTATION OF ACUTE RENAL FAILURE IS OLIGURIA.

5. THE MOST COMMON COMPLICATION OF PERITONEAL DIALYSIS IS ABDOMINAL DISCOMFORT AND PAIN.

6. NO INCIDENCE OF PERFORATION OF GUT IN THIS STUDY.

7. INTERMITTENT PERITONEAL DIALYSIS IS RELATIVELY LESS COSTLY, REQUIRES NO NEED OF CONSTANT ATTENDANCE OF TRAINED PROFESSIONAL AND TECHNICAL PERSONNEL.

8. IN THIS SERIES THE INTERMITTENT PERITONEAL DIALYSIS HAS PROVED TO BE READILY AVAILABLE, SIMPLE, SAFE, AND AN EFFECTIVE ADJUNCT TO THE TREATMENT OF PATIENTS

SUFFERING FROM ACUTE RENAL FAILURE.

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PERFORMA

EVALUATION OF INTERMITTENT PERITONEAL DIALYSIS IN THE MANAGEMENT OF RENAL ACUTE FAILURE

Name: Age: Sex: Occupation: Income: IPNo:
Address:
D O A: Unit: Case No:
D O D: Ward:

H/O PRESENT ILLNESS:

HO Anuria	HO Hiccup	HO Snake Bite
HO Oliguria	HO Convulsion	HO Diarrhoea
HO Hematura	HO Loss of consciousness	HO Vomiting
HO Swelling of Leg	HO Hematemesis	HO Eclampsia
HO Puffiness of face	HO Septic Abortion	HO External Haemorrhage
HO Difficulty in breathing	HO Peripartum Haemorrhage	HO Melena

PAST HISTORY

HO Hypertension	HO D.M.	HO Pharynitis
HO Nephrotoxic Drug Intake	Similar Previous Episodes	HO Impetigo
HO Native Madicine Intake		

CLINICAL EXAMINIATION

Weight:	Hydration:	PR:	BP:	Cellutitis:	Acidotic Breathing:
Pallor:	Jaundior:	Level of Consciousness:		Skin Rashes:	Pedal Edema:
Abdomen:		CVS:	RS:	CNS:	

INVESTIGATION

1. Urine:	Alb:	Sug	Dep			
2. Blood	HB:	TC:	DC:	RBC:		
3. Clotting Time:	4. ECG:	5. X-Ray Chest PA View		6. X-Ray KUB Region	7. USG ABD	

	I IPD					II IPD						III IPD			
BI Sugar															
BI Urea															
Sr. Creatinine															
Sr. Electrolytes															
Sodium															
Potassium															
Urine Output															

COMPLICATIONS

Peritonitis:
a. Symptums:
b. Cell Count:
c. Culture and Sensitivity:

Hemorrage:
(Slight/Sever)
Abdominal Pain

Blockage of Catheter
Perfection of the gut

PATIENTS: IMPROVED: **TAKEN FOR HD:** **WORSEN:** **DIED:**

IPD
No of Exchanges
Indwelling Time
Ironotics
Hypertensin
With or Without Heparin
No of PD.
No Of Days of Improvement

MASTER TABLE

S No.	Name	Age/Sex	Cause of ARF	Oliguria / Anuria	Haematuria	Metbolic Acidosis	Purmonary Oedema	Urine Output
1	Ashokan	25/M	AGE	+	-	-	-	200
2	Kajamithen	65/M	AGE	+	-	-	-	250
3	Kumar	42/M	SB	+	-	-	-	50
4	Subramani	40/M	SB	+	-	-	-	100
5	Kanagaraj	35/M	SB	+	-	-	-	100
6	Hamsha	40/F	SB	+	-	-	-	200
7	Uma Shankar	15/M	SB	+	-	-	-	200
8	Annamal	65/F	AGE	+	-	-	-	200
9	Jhonson	27/M	AGN	+	-	-	-	200
10	Nagaraj	38/M	AGE	+	-	-	-	150
11	Prabhu	17/M	AGE	+	-	+	-	Nil
12	Guna Pakiam	60/F	AGE	+	-	-	-	350
13	Kupavu	50/F	Malaria	+	+	-	-	100
14	Syed Mustaf	27/M	CuSo4	+	-	-	-	250
15	Senthil	25/M	DI	+	-	-	-	150
16	Manikam	19/M	CuSo4	+	+	-	-	200
17	Venkatesan	25/M	CuSo4	+	+	-	-	100
18	Gopal	25/M	CuSo4	+	+	-	-	350
19	Elanchzhiyan	26/M	Sepeticemia	+	-	-	-	250
20	Rajagopal	48/M	AGE	+	-	-	-	100
21	Makala	27/F	PS	+	-	-	-	300
22	Shamugathai	60/F	AGE	+	-	-	-	200
23	Marimuthu	33/M	AGN	+	-	+	-	100
24	Usha	26/F	Obstetric	+	-	-	-	200
25	Shanthanam	37/M	Malaria	+	-	-	-	250

USG/Abdomen

S No.	Name	Blood Sugar	Blood Urea	Creatinine	Potasium	CT	ECG	Right	Left	No Of PD
1	Ashokan	128	128	10.2	4.9	Normal	Normal	Normal	Normal	3
2	Kajamithen	120	78	4.8	4.7	Normal	Normal	Normal	Normal	1
3	Kumar	98	162	8.3	4.9	Normal	Normal	Normal	Normal	1
4	Subramani	90	102	5.2	5.5	Normal	Normal	Normal	Normal	1
5	Kanagaraj	100	80	3.6	5.2	Normal	Normal	Normal	Normal	1
6	Hamsha	80	135	7.8	5.7	Normal	Normal	Normal	Normal	1
7	Uma Shankar	135	130	5.2	5.5	Normal	Normal	Normal	Normal	1
8	Annamal	135	92	4.7	6.1	Normal	Normal	Normal	Normal	1
9	Jhonson	129	114	4.1	3.9	Normal	Normal	Normal	Normal	1
10	Nagaraj	136	62	4.5	4.7	Normal	Normal	Normal	Normal	1
11	Prabhu	124	148	4.9	5.2	Normal	Normal	Normal	Normal	1
12	Guna Pakiam	92	76	3.3	3.7	Normal	Normal	Normal	Normal	1
13	Kupavu	124	68	2.7	3.6	Normal	Normal	Normal	Normal	2
14	Syed Mustaf	98	96	3.8	4.9	Normal	Normal	Normal	Normal	1
15	Senthil	124	130	8	5.1	Normal	Normal	Normal	Normal	1
16	Manikam	100	128	9	5.6	Prolonged	Normal	Normal	Normal	1
17	Venkatesan	114	120	4.6	4.3	Normal	Normal	Normal	Normal	1
18	Gopal	110	120	6.3	5.1	Normal	Normal	Normal	Normal	1
19	Elanchzhiyan	78	106	4.5	5.6	Normal	Normal	Normal	Normal	1
20	Rajagopal	140	104	4.7	5.1	Normal	Normal	Normal	Normal	2
21	Makala	122	84	4.8	5.3	Normal	Normal	Normal	Normal	1
22	Shamugathai	120	120	8	4.9	Normal	Normal	Normal	Normal	1
23	Marimuthu	114	110	6.6	4.5	Normal	Normal	Normal	Normal	2
24	Usha	90	126	5.3	4.7	Normal	Normal	Normal	Normal	1
25	Shanthanam	130	126	5.3	4.6	Normal	Normal	Normal	Normal	1

				Haemorrhage			At Discharge			
S No.	**Name**	**Abdominal Pain**	**Catheter Block**	**Slight**	**Severe**	**Peritonitis**	**Urine Output**	**Urea**	**Creatinine**	**OutCome**
1	Nil	Nil	Nil	Nil	Nil	Nil	1900	37	1.1	Improved
2	Kajamithen	+	-	-	-	Nil	2900	42	1.4	Improved
3	Kumar	+	+	-	-	Nil	2000	38	1.1	Improved
4	Subramani	-	-	-	-	Nil	1850	38	1.6	Improved
5	Kanagaraj	+	+	-	+	Nil	1900	38	1.7	Improved
6	Hamsha	+	-	-	-	Nil	1800	38	1.6	Improved
7	Uma Shankar	+	+	-	-	Nil	2000	38	1.2	Improved
8	Annamal	-	-	-	-	Nil	2000	30	1.7	Improved
9	Jhonson	+	+	-	+	Nil				Expired
10	Nagaraj	+	+	-	-	Nil	1400	30	1.2	Improved
11	Prabhu	-	+	-	-	Nil	1400	36	1.8	Improved
12	Guna Pakiam	+	-	-	-	Nil	1300	40	1.6	Improved
13	Kupavu	+	-	-	-	Nil	1800	40	2	Improved
14	Syed Mustaf	+	-	-	-	Nil	1800	38	1.2	Improved
15	Senthil	+	+	-	+	Nil				Started HD
16	Manikam	+	+	+	-	Nil				Expired
17	Venkatesan	+	+	+	-	Nil	2200	34	1.3	Improved
18	Gopal	-	+	+	-	Nil				Started HD
19	Elanchzhiyan	+	-	-	-	Nil	1500	30	1.1	Improved
20	Rajagopal	+	-	-	-	Nil	1600	26	1.3	Improved
21	Makala	+	-	-	-	Nil	2200	30	0.9	Improved
22	Shamugathai	+	-	-	-	Nil				Expired
23	Marimuthu	+	-	-	-	Nil				Expired
24	Usha	-	-	-	-	Nil	1450	24	0.9	Improved
25	Shanthanam	+	-	-	-	Nil				Expired

S No.	Name	Age/Sex	Cause of ARF	Oliguria / Anuria	Haematuria	Metbolic Acidosis	Purmonary Oedema	Urine Output
26	Jayasing	47/M	AGE	+	-	-	-	300
27	Kasinathan	42/M	AGE	+	-	-	-	150
28	Pandian	48/M	LEP	+	-	+	-	100
29	Ramaiyah	50/M	AGE	+	-	+	-	150
30	Annadurai	53/M	AGE	+	-	-	-	200
31	Srinivasan	35/M	LEP	+	+	-	-	150
32	Velautham	45/M	AGE	+	-	-	-	150
33	Thilagath Begam	45/F	LEP	+	-	-	-	300
34	Malarkodi	45/F	AGE	+	-	-	-	50
35	Lakshmanaiah	36/M	CuSo4	+	-	-	-	200
36	Ramalingam	55/M	AGE	+	-	-	-	200
37	Senthil Kumar	20/M	AGN	+	+	-	-	100
38	Rajendran	50/M	Sepeticemia	+	+	-	-	150
39	Jayanthi	38/F	AGE	+	-	-	-	750
40	Velmurugan	32/M	AGE	+	-	-	-	200
41	Kaniyappan	35/M	AGE	+	-	+	-	200
42	Shajagan	48/M	Sepeticemia	+	-	-	-	200
43	Magesh	31/M	AGE	+	-	+	-	100
44	Meena	43/F	LEP	+	-	-	-	150
45	Shalini	37/F	AGE	+	-	-	-	450
46	Ramesh	29/M	AGN	+	-	-	-	100
47	Kannan	50/M	AGE	+	-	-	-	200
48	Shanthi	37/F	CuSo4	+	-	-	-	300
49	Panjanathan	50/M	AGE	+	-	-	-	250
50	Dhayalan	37/M	CuSo4	+	-	-	-	250

								USG/Abdomen		
S No.	**Name**	**Blood Sugar**	**Blood Urea**	**Creatinine**	**Potasium**	**CT**	**ECG**	**Right**	**Left**	**No Of PD**
26	Jayasing	87	74	6.6	4.5	Normal	Normal	Normal	Normal	3
27	Kasinathan	92	64	3.1	5.6	Normal	Normal	Normal	Normal	1
28	Pandian	112	126	4.3	4.8	Normal	Normal	Normal	Normal	1
29	Ramaiyah	110	80	6.9	4.8	Normal	Normal	Normal	Normal	2
30	Annadurai	135	66	5.6	4.6	Normal	Normal	Normal	Normal	1
31	Srinivasan	85	120	5.9	4.6	Normal	Normal	Normal	Normal	1
32	Velautham	85	86	5.3	4.5	Normal	Normal	Normal	Normal	1
33	Thilagath Begam	123	130	11.3	5.1	Normal	Normal	Normal	Normal	2
34	Malarkodi	134	90	8.7	5.1	Normal	Normal	Normal	Normal	2
35	Lakshmanaiah	98	128	6.5	5.5	Normal	Normal	Normal	Normal	1
36	Ramalingam	134	96	7.9	4.8	Normal	Normal	Normal	Normal	2
37	Senthil Kumar	86	94	4.8	6.1	Normal	Normal	Normal	Normal	1
38	Rajendran	124	85	5.9	5	Normal	Normal	Normal	Normal	2
39	Jayanthi	83	110	7.1	4.5	Normal	Normal	Normal	Normal	1
40	Velmurugan	93	70	4.2	5.3	Normal	Normal	Normal	Normal	2
41	Kaniyappan	127	75	9	5.1	Normal	Normal	Normal	Normal	1
42	Shajagan	133	130	6.4	5.3	Normal	Normal	Normal	Normal	2
43	Magesh	86	146	8.2	5.1	Normal	Normal	Normal	Normal	1
44	Meena	132	130	7.4	4.5	Normal	Normal	Normal	Normal	2
45	Shalini	95	75	2.3	5.2	Normal	Normal	Normal	Normal	1
46	Ramesh	100	106	6.4	3.4	Normal	Normal	Normal	Normal	2
47	Kannan	110	80	6.4	4.1	Normal	Normal	Normal	Normal	2
48	Shanthi	92	60	4.1	4.9	Normal	Normal	Normal	Normal	2
49	Panjanathan	94	88	11.7	4.9	Normal	Normal	Normal	Normal	1
50	Dhayalan	120	126	6.8	5.9	Normal	Normal	Normal	Normal	1

				Haemarrhage		**At Discharge**	

S No.	Name	Abdominal Pain	Catheter Block	Slight	Severe	Peritonitis	Urine Output	Urea	Creatinine	OutCome
26	Jayasing	+	-	-	-	Nil	1750	36	1.6	Improved
27	Kasinathan	+	-	-	-	Nil	2400	32	1.4	Improved
28	Pandian	+	+	+	-	Nil				Started HD
29	Ramaiyah	+	-	-	-	Nil	2100	38	1.6	Improved
30	Annadurai	+	-	-	-	Nil	1800	38	1.7	Improved
31	Srinivasan	+	-	-	-	Nil				Started HD
32	Velautham	-	-	-	-	Nil	1800	42	1.9	Improved
33	Thilagath Begam	+	+	-	-	Nil	1600	32	1.6	Improved
34	Malarkodi	+	+	-	-	Nil	1800	36	1.8	Improved
35	Lakshmanaiah	-	-	-	-	Nil	1600	30	1.4	Improved
36	Ramalingam	+	-	-	-	Nil				Expired
37	Senthil Kumar	-	-	-	-	Nil				Expired
38	Rajendran	-	-	-	-	Nil				Expired
39	Jayanthi	+	-	-	-	Nil	1900	34	1.4	Improved
40	Velmurugan	-	-	-	-	Nil	1800	34	1.8	Improved
41	Kaniyappan	-	-	-	-	Nil	2400	37	1.9	Improved
42	Shajagan	-	-	-	-	Nil	1900	38	1.2	Improved
43	Magesh	-	-	-	-	Nil				Expired
44	Meena	-	-	-	-	Nil	2100	30	1.4	Improved
45	Shalini	+	-	-	-	Nil	1100	38	1.7	Improved
46	Ramesh	-	-	-	-	Nil	1900	36	1.6	Improved
47	Kannan	-	-	-	-	Nil	2300	40	1.5	Improved
48	Shanthi	-	-	-	-	Nil	2200	41	1.4	Improved
49	Panjanathan	-	-	-	-	Nil				Started HD
50	Dhayalan	-	+	+	-	Nil				Started HD

S No.	Name	Age/Sex	Cause of ARF	Oliguria / Anuria	Haematuria	Metbolic Acidosis	Purmonary Oedema	Urine Output
51	Makilan	31/M	AGE	+	-	-	-	50
52	Dilagath	37/F	Obstetric	+	-	+	-	250
53	Tharman	37/M	CuSo4	+	-	-	-	250
54	Vembuliraja	37/M	CuSo4	+	-	-	-	125
55	Raman	37/M	AGE	+	-	-	-	250
56	Kanniyamal	60/F	LEP	+	+	-	-	250
57	Babu	34/M	AGE	+	+	-	-	100
58	Ashlam Basha	32/M	CuSo4	+	+	-	-	50
59	Narashiman	27/M	DI	+	+	-	-	100
60	Mohan	37/M	CuSo4	+	+	-	-	200

								USG/Abdomen		
S No.	**Name**	**Blood Sugar**	**Blood Urea**	**Creatinine**	**Potasium**	**CT**	**ECG**	**Right**	**Left**	**No Of PD**
51	Makilan	82	138	8.9	5.4	Normal	Normal	Normal	Normal	2
52	Dilagath	97	92	5.2	4.9	Normal	Normal	Normal	Normal	1
53	Tharman	130	146	7.7	4.4	Normal	Normal	Normal	Normal	1
54	Vembuliraja	127	69	3.2	4.8	Normal	Normal	Normal	Normal	2
55	Raman	114	148	7.7	5.4	Normal	Normal	Normal	Normal	1
56	Kanniyamal	107	160	6.6	3.1	Normal	Normal	Normal	Normal	2
57	Babu	75	130	7.6	7.2	Normal	Normal	Normal	Normal	1
58	Ashlam Basha	77	150	9.6	5.4	Normal	Normal	Normal	Normal	2
59	Narashiman	97	140	6.2	5.6	Normal	Normal	Normal	Normal	2
60	Mohan	104	130	7.8	5.6	Normal	Normal	Normal	Normal	2

				Haemarrhage			At Discharge			
S No.	Name	Abdominal Pain	Catheter Block	Slight	Severe	Peritonitis	Urine Output	Urea	Creatinine	OutCome
51	Makilan	-	-	-	-	Nil	2200	36	1.4	Improved
52	Dilagath	+	-	-	-	Nil	1800	40	1.6	Improved
53	Tharman	-	-	-	-	Nil	2000	34	1.4	Improved
54	Vembuliraja	-	-	-	-	Nil	2100	36	1.6	Improved
55	Raman	-	-	-	-	Nil	2000	34	1.4	Improved
56	Kanniyamal	-	+	+	-	Nil	1800	30	1.2	Improved
57	Babu	-	-	-	-	Nil	1900	32	1.2	Improved
58	Ashlam Basha	+	-	-	-	Nil				Started HD
59	Narashiman	-	-	-	-	Nil	2100	28	0.9	Improved
60	Mohan	+	-	-	-	Nil	1700	32	1.4	Improved

ABBREVIATIONS

AGE – Acute Gastro Enteritis
SB – Snake Bite
AGN – Acute Glomerulo Nephritis
$CuSO_4$ – Copper Sulphate
CT – Clotting Time
ECG – Electro Cardiogram
LEP – Leptospirosis
USG – Ultrasonogram